The
VETERINARY RECORD
Founded by William Hunting, F.R.C.V.S., in 1888
7, MANSFIELD STREET, PORTLAND PLACE, LONDON, W.1. MUSEUM 6541
No. 44 OCTOBER 29th, 1955 Vol. 67
Registered for Transmission as a Newspaper *Price 2s. post free*

The Queen at Cambridge

Opening of New School of Veterinary Medicine

THE Queen, accompanied by the Duke of Edinburgh, visited Cambridge on October 20th for the purpose of opening the new School of Veterinary Medicine. Her Majesty and the Duke were received at the station by the Lord Lieutenant of the County, Captain R. G. Briscoe, and the Mayor, Alderman E. T. Halnan. At the Guildhall the Guard of Honour of No. 629 Airborne Light Regiment R.A. (T.A.) presented arms.

A most distinguished academic, professional and official company was present, including many professors of Cambridge, and other universities, the President and Registrar of the Royal College of Veterinary Surgeons, and Sir Thomas Dalling. Sir Alan Hitchman represented the Minister of Agriculture, who was unable to be present.

The Royal visitors arrived at the School at 2.45 p.m. when the Chancellor of the University, Lord Tedder, presented Professor Sir Lionel and Lady Whitby, Professor and Mrs. W. I. B. Beveridge, Professor and Mrs. L. P. Pugh, and Sir Alan and Lady Hitchman. With the Vice-Chancellor he then conducted the Royal party up the south steps to their seats in the portico.

Lord Tedder then made an address of welcome in which he said:

" May it please Your Majesty, it is my very great privilege this afternoon to welcome you and His Royal Highness the Duke of Edinburgh to the University of Cambridge. His Highness has already honoured the University by more than one visit and I rejoice to see he is this day wearing the robes of the Doctorate which he consented to receive when he opened the new building of the engineering laboratories in 1952. You, Your Majesty, are paying your first visit, not only to the University but to the City of Cambridge, and members of the University, young and old, have already joined the citizens in showing their warm appreciation of the great honour you have done us, and I can assure you that should you be pleased to visit Cambridge again, however soon, your welcome will be equally warm.

" On this occasion you have graciously consented to open these buildings here which the University has been encouraged and enabled by Your Majesty's government to erect for the training of veterinary surgeons and for the furtherance of the study of Veterinary Science. The Cambridge School of Veterinary Medicine is the youngest of the six which are now established within the Universities of Great Britain, but the desirability of founding a place for veterinary studies has been in our minds for many years. Sir Clifford Allbutt, who, throughout his tenure as Regius Professor of Physic from 1892 to 1925, championed the cause of comparative pathology, advocated raprochement of the studies relating to the association of man and animal and applied for the investigations of the diseases of animals to throw light on those of man. A professorship of Animal Pathology endowed by the Ministry of Agriculture and Fisheries was established in 1923 but it was not until the recommendation of the second report of the Loveday Committee had been accepted by the Ministry of Agriculture and the University that the way was open for adding to the Department of Animal Pathology a Department of Veterinary Clinical Studies and so creating in the University a full School of Veterinary Medicine. The first veterinary students entered the University in 1949 and, after completing the six years' course, received the new degree of Bachelor of Veterinary Medicine in June, 1955. The instruction of these students and of those of some later years has been handicapped by lack of proper accommodation and the work on these buildings which you have consented to open to-day was not started until the summer of 1950 and was not completed until this summer. The inherent difficulties were, however, overcome and we are all the more confident that the School now properly equipped will flourish and we feel that there are good reasons for our confidence.

" The intellectual atmosphere of Cambridge is, we feel, calculated to ensure the breadth of knowledge and of interest which is the foundation of civilised

Her Majesty the Queen, watched by His Royal Highness the Duke of Edinburgh, makes the first signature in the Visitors' Book at the School of Veterinary Medicine. In the background is the painting of the Queen's racehorse, "Aureole," which was presented to Her Majesty. On the left of the picture is Professor L. P. Pugh, and beside him, Professor W. I. B. Beveridge.

society. The veterinary student here will now find in Cambridge strong schools of medicine and of agriculture, subjects to which his future will be linked, and a student who has passed the three years, undergraduate years, in the company of young people whose interests range from various sciences to the arts, and who include representatives of many countries other than his own, must have learnt something of the purpose of life and its diversities. His entry into the three subsequent years of Veterinary Clinical Studies to be carried out in these buildings will therefore have been prepared against the best of academic backgrounds and he will realise that the acquisition of technical knowledge is not an end in itself but an equipment for service and that in devoting his life to the service of animals he will serve not only animals but also man.

"Your Majesty, may I now humbly beg leave to ask you to open the buildings of the Cambridge School of Veterinary Medicine."

Replying to the Chancellor, Her Majesty spoke as follows:

"I thank you for the kind welcome with which you have received me and my husband. My father had the happiest memories of the time he spent here and it is a real pleasure to me to visit his University and to continue the long association of my family with Cambridge. The opening of these new buildings will crown the effort of many years spent in the cause of veterinary medicine, together with the recent institution of a faculty here in the study of this branch of medical science. It holds promise of further achievements in the highest tradition of Cambridge scholarship.

"Cambridge is famed for many causes and not least among them for its splendid distinction in advancing the frontiers of scientific knowledge. Many years ago, Sir Isaac Newton, one of your greatest men, spoke with humility of the great ocean of truth which lay all undiscovered before him. Later generations of Cambridge men and women have achieved much in its exploration but, with that same spirit of humility, we must acknowledge that there is still much to be sought. Every advance in knowledge

discloses new problems awaiting our enquiry and to meet that challenge we often need new facilities for study and research and new buildings such as these.

"I must confess to you that, as a lover of animals, I have often thought with concern that veterinary medicine was the Cinderella of the sciences and that insufficient time and energy were devoted to it; but now your new course of study in the nature of animals and the ills which beset them can take its proper place beside other sciences in the humane and liberal atmosphere of this University. It is surely right that it should be associated with the other medical studies if it has a direct influence on the well-being of man, for throughout the world there are peoples whose futures must depend on the better development of age-old methods of agriculture and animal husbandry. A responsibility rests on us and other nations to help those less fortunate and less skilled and we look to veterinary medicine to play an important part in the solution of the urgent problems that confront us.

"The extension of opportunities for this study does more therefore than satisfy the wishes of those people who, like myself, have a deep regard for the welfare of animals. It encourages research which may be of the greatest moment to mankind. I congratulate all those who have shared in the planning of these buildings, in their construction and in their equipment, and I declare them open in the confident hope that the work done in them will worthily serve the needs of every living thing that moves upon the earth in the service of man."

The Chancellor then invited the Queen and the Duke to enter the building. He accompanied the Queen and the Duke into the main entrance hall, and there asked Professor Beveridge and Professor

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The Queen watching a demonstration of the electrocardiograph which was presented to her on behalf of the City of Cambridge by the Mayor, Alderman E. T. Halnan. Professor L. P. Pugh and Professor W. I. B. Beveridge are also present.

The main building of the Cambridge University School of Veterinary Medicine which has been designed by Ian Forbes, F.R.I.B.A.

Some Factors in the Epidemiology of Parasitic Bronchitis in Cattle

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ONE of the outstanding problems of parasitic bronchitis in cattle is the way in which the causal organism, *Dictyocaulus viviparus*, persists from year to year on a farm. Four possible mechanisms are:—

(1) the larvae survive on the pasture over the winter.

(2) adult lungworms persist in the bronchi of cattle from autumn until the following spring.

(3) infective larvae ingested in the autumn by a relatively immune animal undergo an inhibition of development which may prolong the prepatent period until the spring.

(4) where stock are outwintered, continued reinfection may occur.

Work carried out in Britain (Michel & Rose, 1954; Soliman, 1952), Germany (Wetzel, 1948), Russia (Orlov, 1946) and America (Porter, 1942) has indicated that bovine lungworm larvae cannot survive on pasture from the autumn until the following spring, the longest period of survival recorded being from October until January, *i.e.* 13 weeks (Michel & Rose, 1954). Wetzel (1948) as a result of periodic faecal examinations of recovered field cases concluded that the survival of the disease from year to year on a farm is wholly dependent on pasture contamination by "silent carriers," *i.e.* bovines which remain subclinically infected from one grazing season until the next. An extension of this theory has been suggested by Taylor (1952) who stated that inhibition of development may ultimately be found to explain the overwintering of husk worms in cattle.

The purpose of this communication is to describe some experiments and observations dealing with these problems which were carried out between 1952 and 1954 in Scotland.

The Survival of Larvae on Pasture

From field observations it appeared to us that a number of outbreaks of parasitic bronchitis were probably initiated by larvae which had persisted on the pasture from the previous autumn. In order to determine if such survival could occur, the following experiments were carried out.

1952-1953

Experiment 1

A plot, 45 square yards in area, was grazed by an infected calf from October 24th until November 4th; during this time the larval output varied between 150 and 50 larvae per gramme of faeces. In addition, from November 17th until November 30th, the faeces of three infected cows were spread on this plot, the larval counts of these animals ranging from 150 to 750 larvae per gramme of faeces.

No animals were grazed on this plot from November 30th until May 14th of the following year when two three-month-old calves were turned on to it, remaining until June 11th. (These two calves and all others used in subsequent experiments for this purpose were reared indoors and fed on milk, hay and concentrates.) At autopsy the lungs of one calf contained six young adult lungworms while the lungs of the other were negative. The larval survival time was five and a half months.

Experiment 2

All of the faeces from a cow which was excreting 200 larvae per gramme, were spread daily on a plot, 80 square yards in area, from December 6th to 12th. The plot was closed until May 10th when two calves were turned out to graze upon it. The faeces of one of the calves was found to contain a small number of larvae, detected by Baermann examination, on July 14th and on the following day 60 larvae per gramme faeces were recovered; thereafter the faeces were negative until July 20th when the calf was killed. No adult lungworms were found at autopsy although lesions were present in the diaphragmatic lobes of both lungs. When the lungs were minced and subjected to Baermann examination, first stage Dictyocaulus larvae were recovered. Histological examination of the lesions showed the presence of first stage larvae in the alveoli (*vide* Jarrett, McIntyre & Urquhart, 1954). The larval survival time was five months.

The second calf was autopsied on July 30th; no lungworms or lesions indicative of lungworm infection were found.

Experiment 3

A plot 800 square yards in area was grazed by five field cases of parasitic bronchitis from July 17th until the middle of August; during this period the faecal counts ranged from 480 larvae per gramme to 50 larvae per gramme. The plot was then closed until the third week of July the following year, when three calves were put on to it. Faecal examinations were not carried out and the calves were autopsied on September 2nd when no adults were found in the bronchi, although there were lesions around the distal parts of the diaphragmatic lobes of all the lungs. On histological examination these lesions were found to contain first-stage larvae of *D. viviparus* indicating the previous presence of gravid adults.

The larval survival time was 11 months.

The plot which was used in the first of these experiments was separated by a single fence from the rest of the field which was grazed intermittently by a few cows; the second and third plots were in fields which were empty during the entire period of the experiment.

1953-1954

In view of the positive results obtained during 1952-1953 a further five plots, each measuring 30 yards by 10 yards, were fenced off in the summer of 1953. These were constructed of pig-link fencing wire and each plot was separated from its neighbour by a strip 2 feet wide. In order to avoid contamination of the plots with faeces from other animals grazing on the outside perimeter of the plots, a four-strand barbed-wire fence was erected around the plots. The general layout is illustrated in Fig. 1.

Six heavily infected field cases of parasitic bronchitis, the faecal larval counts of which ranged from 500 to 2,000 larvae per gramme were turned on to four of these plots during August and September of 1953 while the fifth plot was manured with faeces from the same animals during the latter half of September. The plots were then closed until the following summer when young calves reared indoors were put on to them.

The results of this experiment are summarised in Table I and show that four out of eight calves on three of the five plots became infected.

A second experiment on larval longevity was carried out in which small masses of faeces, containing relatively large numbers of larvae (*v.* Table II) were placed on grass grown in earthenware pots; the soil was sterilised before the grass seeds were sown. The pots were kept on the roof of a building and after three months the faeces and grass were removed from a pot, suspended in water and fed to a calf; the surface of the soil in the pot was rinsed with water and this, too, was fed to the calf. This was repeated when the faeces were six, nine and 12 months old. The results of this experiment are detailed in Table II and show that the three-, six- and nine-month infections were positive although the three 12-month attempts were negative. These calves were reared indoors, bedded on sawdust, and were fed only on milk throughout their lives, so as to eliminate the possibility of extraneous infection.

The temperature and rainfall recorded in this district during the two years over which these experiments were conducted are shown in Table III.

The Incidence of Carriers

Efforts to determine the incidence of carriers were directed along three lines in 1953-1954.

(1) Eighteen calves, aged six to nine months, in each of which clinical parasitic bronchitis was confirmed by faecal examination during the summer and early autumn of 1953, were purchased and kept indoors in a loose box or byre at the Veterinary Hospital over the winter. Regular faecal examination carried out on these animals during the winter and spring gave consistently negative results. These stirks were killed and autopsied at the times stated in Table IV. In each case the lungs were removed and the bronchi examined for lungworms; scrapings of the bronchial mucus were mixed with saline and examined for immature worms, and the lungs were minced and subjected to Baermann examination for the same purpose.

FIG. 1.—This illustrates the general layout of the plots used in the larval longevity experiments during 1953-54.

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FIG. 1.—This illustrates the general layout of the plots used in the larval longevity experiments during 1953-54.

TABLE I

SURVIVAL OF *D. viviparus* LARVAE ON PASTURE 1953–1954

Plot number	Last grazed by infected animal	Grazed by susceptible calves	Number of calves	Results	Survival time of larvae
1	9.9.53	4.6.54 to 1.9.54	1	Larvae in faeces (Baerman technique) on eight occasions from 13.7.54 to 2.8.54 ; faeces negative thereafter. Coughed from 12.7.54 until killed (1.9.54). No lungworms found but typical lesions present.	9 months
2	9.9.53	4.6.54 to 1.9.54	1	Coughing and tachypnoea noted 31.8.54. Twelve lungworms and pneumonia found at autopsy (1.9.54)	9 months
3	Manured with infected faeces during latter part of September, 1953	4.7.54 to 15.9.54	2	Neither lungworms nor lesions found.	—
4	10.9.53	2.9.54 to 6.10.54	2	1. Two lungworms and typical lesions present. 2. Nine lungworms and typical lesions present.	12 months
5	1.9.53	31.8.54 to 6.10.54	2	Neither lungworms nor lesions found.	—

TABLE II

SURVIVAL OF *D. viviparus* LARVAE IN POTS OF GRASS

Calf number	Age of faeces months	Weight of original faeces	Number larvae/gm. in original faeces	Number of lungworms obtained from calf at autopsy
*1	3 (July–Oct.)	60 gm.	400	28
*2	6 (Aug.–Feb.)	60 gm.†	1,100	19
*3	9 (Aug.–May)	105 gm.†	4,000	18
4	12 (Oct.–Oct.)	170 gm.†	1,850	No lungworms present
5	12 (Oct.–Oct.)	170 gm.†	1,850	No lungworms present
6	12 (Oct.–Oct.)	170 gm.†	1,850	No lungworms present

* In calves 1, 2 and 3, during the later stages of the infection, symptoms of coughing and tachypnoea were noted.

† The 6, 9 and 12 month old faeces had been exposed to severe frost.

From the results recorded in Table IV, it will be seen that adult lungworms were detected in small numbers in only one of the 18 recovered cases; immature worms were not found in any of the lungs. The animal which contained adult worms had been taken indoors when affected with parasitic bronchitis during the previous September and had been tied by the head in a byre until the time of death; the worms, therefore, must have persisted in the lungs for eight months as it is unlikely that reinfection occurred during this period.

(2) Farms where outbreaks were investigated by us during the summer and autumn of 1953 were visited during the following winter and spring and faecal samples were obtained from recovered cases. The faeces were examined by McMaster and Baermann techniques on each occasion. In all, the faeces of 63 stirks which were wintered indoors on eight farms were examined each month until May and in only two cases were positive results obtained. In one, the faeces were shown by Baermann examination to contain a small number of larvae in April and in the other, from a different farm, counts of 30 and 50 larvae per gramme were recorded on two occasions in April. This latter animal was brought to the Veterinary Hospital, the faecal count dropped to zero and at autopsy on May 21st no worms were found although there were pneumonic lesions at the posterior tip of each diaphragmatic lobe.

(3) A total of 493 bovine lungs were examined for the presence of lungworms between the beginning of February and the end of May 1954, and of this total 361 were from adults and 132 were from stirks (yearlings). The monthly incidence of infected lungs, recorded as a percentage of the total number of cow and stirk lungs examined is shown in Table V.

Discussion

The first set of experiments described above show conclusively that larvae of *D. viviparus* can survive on pasture for a year (see Table I). Over a period of two years, 15 calves were grazed on seven plots at varying intervals after each plot had been contaminated in the preceding grazing season with faeces containing lungworm larvae; nine calves grazing five plots became infected with *D. viviparus*.

These findings conflict with those of other workers (*v.* introduction) and may be explained in several ways. The climatic conditions in the west of Scotland may be conducive to the prolonged survival of larvae; we have appended a table (Table III) summarising the relevant meteorological data for this area. An alternative explanation may depend upon the method which we have adopted for the recovery of larvae from the pasture; this consisted of grazing young susceptible animals on plots known to have been previously contaminated with faeces containing large numbers of larvae, killing these calves after a suitable time and subjecting their lungs to detailed parasitological and histological examination. We consider that this method of biological recovery under controlled conditions affords the only permissible criterion by which the maximum survival time of infective

TABLE III

METEOROLOGICAL DATA FOR PERIOD OF EXPERIMENTS (SUPPLIED BY METEOROLOGICAL OFFICE, CIVIL AIRPORT, RENFREW)

	Year	Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
Mean Screen Temperature Monthly $\frac{\text{Max} + \text{Min.}}{2}$	1952	—	—	—	—	—	—	59·4	58·2	51·0	46·9	38·3	37·5
„ „	1953	40·0	41·6	40·9	43·7	54·0	58·1	58·3	58·7	56·4	50·0	47·1	43·1
„ „	1954	38·7	36·5	41·2	45·7	52·3	55·2	55·8	56·5	52·3	50·1	—	—
Top figure ; Number of occasions of ground frost (*i.e.*, Grass min. 30°F. or less, period 9 p.m. to 9 a.m.)	1952	—	—	—	—	—	—	0 31°F.	1 30°F.	6 23°F.	10 16°F.	18 10°F.	17 07°F.
	1953	12 09°F.	13 12°F.	18 16°F.	16 11°F.	2 26°F.	1 30°F.	0 32°F.	1 28°F.	1 30°F.	7 20°F.	7 22°F.	11 19°F.
Bottom figure ; Lowest grass minimum temperature in month	1954	14 14°F.	11 17°F.	8 13°F.	13 19°F.	3 28°F.	1 28°F.	3 27°F.	2 29°F.	6 20°F.	7 21°F.	—	—
Total monthly rainfall in inches	1952	—	—	—	—	—	—	2·07	3·07	3·02	4·39	1·87	3·76
„ „	1953	1·19	1·45	0·98	1·43	1·91	2·44	4·05	3·12	3·85	2·49	5·86	4·78
„ „	1954	5·14	4·09	2·33	1·47	3·22	2·54	2·65	5·09	4·70	8·75	—	—
30 year monthly mean	—	4·64	3·07	2·45	2·46	2·61	2·36	3·20	3·37	3·70	4·73	4·40	4·12

TABLE IV

THE NUMBER OF LUNGWORMS FOUND IN RECOVERED CASES OF PARASITIC BRONCHITIS

Number of animals	Date when ill	Date of autopsy	Number of lungworms found at autopsy
1	August 1953	Sept. 1953	0
2	August 1953	Nov. 1953	0
1	August 1953	Jan. 1954	0
13	August 1953	May 1954	0
1	Sept. 1953	May 1954	6 Adults

TABLE V

THE INCIDENCE OF CARRIERS IN KNACKERY SURVEY

Month	Adults			Stirks		
	Total	Positive	Per cent. Positive	Total	Positive	Per cent. Positive
February	84	2	2·4	42	3	7·1
March	93	3	3·2	34	5	14·7
April	83	4	4·8	26	8	30·7
May	101	11	10·8	30	6	20·0

larvae may be determined. The conclusions of Wetzel (1948) depended on clinical findings and faecal examinations in a herd in which the disease was known to have occurred while Michel & Rose (1954) used a technique in which larvae were recovered directly from grass, soil and faeces from small artificially infected boxes of grass. Porter (1942) used a technique similar to ours and concluded that the larvae survived for one week but not for six or seven; these experiments were carried out in Alabama, U.S.A. in two years when the temperature during the period of experiment ranged from 60° to 104° F. in the first year to 47° to 97° F. in the second. From the same period (April to September) in our experiments, the lowest temperature recorded was 11° F. (grass temperature) and the monthly mean screen temperatures ranged from 43·7° to 59·4° F. From these marked differences in temperature, it is not surprising that the results obtained by Porter and by us differ since desiccation probably has a marked effect on larval longevity.

The numbers of lungworms which we recovered from the calves infected on the contaminated plots were in all cases small and it is difficult to assess the importance of this degree of survival in the epidemiology of the majority of field outbreaks. However, two factors must be borne in mind; first, the plots did not have a greater degree of contamination than is often found in the field; and secondly, the possible importance of an initial light infection at the beginning of a grazing season in initiating a subsequent outbreak cannot be ignored. Furthermore, from field observations we suggest that on a number of occasions, the survival of larvae from one grazing season to the next has been responsible for outbreaks of the disease, *e.g.* on two occasions, outbreaks occurred in young calves on new pasture which had not been grazed by bovines during the preceding year, but had been manured during the autumn and winter.

One criticism of our experiments might be that the diet of the calves which we used contained unsterilised hay and that infection may have been derived from this source; this is unlikely since of many calves

reared for other experiments and fed on the same hay, none showed lungworms at autopsy. In addition the calves used to determine the survival time of larvae in small pots of grass (longest survival recorded, nine months) were fed only on milk and bedded on sawdust throughout their entire lives.

The evidence incriminating carriers as a potential source of infection has been obtained by us in several different ways. Only one out of 18 young cattle which had recovered from severe parasitic bronchitis the preceding autumn was found to be harbouring lungworms (six in number) at autopsy in May. The faecal counts of all, after recovery from the primary illness, were consistently negative. A similar small proportion of carriers, as judged by faecal larval counts, was detected in a follow up, in commercial herds, of animals which had suffered from severe husk. A much higher incidence of infected stirks (*v.* Table V), was found in the knackery survey and this may be accounted for in several ways. All of the animals in the hospital and farm series were known to have had severe infestations and may have acquired such a degree of immunity as to preclude their acting as carriers while it is possible that many of the knackered animals had been lightly infected during the grazing season and so had been able to continue harbouring parasites. It is also probable that many of these latter animals were wintered out of doors and so may have been infected a short time before being examined although we obtained definite evidence that some of the knackery cases had been tied up by the head all winter.

A recent finding is of interest in this connection. The scope of the survey has now been extended to include a second knackery of Ayrshire dealing almost exclusively with bovines from intensive dairy herds. In February, 1955, we found that two of 80 adult lungs (2.5 per cent.) and 13 of 42 stirk lungs (31 per cent.) contained lungworms whereas in the knackery where the former survey was carried out, two of 62 adult lungs (3.2 per cent.) and four of 27 stirk lungs (14.8 per cent.) were affected during the same period. The difference in numbers of carriers in these two knackeries may reflect the higher incidence of the disease in an area which is predominantly concerned with intensive dairy farming. It must be pointed out that the great majority of these carriers were about one year old; that their lungs contained only small numbers (five to 20) of lungworms; and that several had been tethered in stalls since the autumn.

The observations described above show that two factors are concerned in the perpetuation of parasitic bronchitis from year to year; first that larvae can survive on the pasture over the winter; and secondly that animals may act as silent carriers over the same period. From this, two important conclusions may be drawn concerning the relationship of grazing management to the prevention of the disease. (1) The disease may be completely prevented in calves by allowing them to graze only on grass which has not before been grazed by cattle or manured during the preceding winter. (2) If such ground is not available the risk of infection will be decreased by preventing the calves from grazing with yearlings or on any field which has been grazed by the latter in the preceding 12 months.

Summary

1. Evidence is presented to show that the larvae of *D. viviparus* may survive and remain infective for one year on pasture in the west of Scotland.

2. The occurrence of carriers of the disease is demonstrated and their numerical and approximate age incidence given.

3. The significance of these findings is discussed with regard to the control of the disease.

Acknowledgments.—This work was carried out with the aid of a grant from the Agricultural Research Council. We wish to thank the Meteorological Officer, Renfrew Civil Airport, for compiling Table III and the many veterinary surgeons who have given us unfailing co-operation.

References

JARRETT, W. F. H., MCINTYRE, W. I. M., & URQUHART, G. M. (1954). *Vet. Rec.* **66.** 665.
MICHEL, J. F., & ROSE, J. H. (1954). *J. comp. Path. and Therap.* **64.** 195.
ORLOV, I. V. (1946). *Veterinarya.* **23.** 4.
PORTER, D. A. (1942). *Proc. helminth. Soc. Wash.* **9.** 60.
SOLIMAN, K. N. (1952). *Brit. Vet. J.* **108.** 167 and 204.
TAYLOR, E. L., B.V.A. Publication No. 23. London 1952.
WETZEL, R. (1948). *Monatsschr. Vet. Med.* **3.** 141.

The Queen at Cambridge—*Continued from page 818*

Pugh to take Her Majesty and the Duke on a short tour of the building. The route followed passed along the north corridor into and through the Parasitology Laboratories back into the corridor and through Teaching Laboratory No. 1 (demonstration on virus pneumonia of pigs) past the Senior Common Room, into and through Teaching Laboratory No. 2, and through adjoining Preparation Room. Mounting to the first floor, the party then passed through the Lecture Theatre (upper part) and back along the corridor, viewing the Library from the gallery.

On returning to the ground floor the Chancellor asked Professor Pugh to conduct Her Majesty into the Hospital. The Vice-Chancellor asked Professor Beveridge to conduct the Duke.

The Professors conducted the Royal guests along the corridor, passing the Clinical Pathology Laboratories on the left and the Post-Mortem and Surgical Anatomy Rooms on the right, reaching the Rotunda, where electro-cardiographic work was demonstrated. Earlier in the day the Mayor had presented to the Queen, as a gift from the City, an electrocardiograph, which the Queen hoped to " put to good use not far from here." Thence through the X-Ray Room to the Large Animal Operating Theatre, where radiographs dealing with orthopaedic work were on view. Next to the Sterilising Room, Small-Animal Operating Theatre (anaesthetic apparatus and bio-physical recorder) and Anaesthetic Room, and finally to the Kennels.

In the main entrance lobby, Professor Pugh then presented to the Queen a portrait of her horse " Aureole," a gift from the Staff. The portrait was painted by the Kentish artist, Miss F. M. Hollans.

The Disintegration of Bull Spermatozoa

BY

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Introduction

THE term "disintegration" was applied to spermatozoa by Chang (1943) to denote their separation into isolated heads and tails. In Guernsey bulls a form of sterility occurs which is characterised by the disintegration of virtually all the ejaculated spermatozoa (Hancock & Rollinson, 1949). Evidence is presented here that in these cases disintegration occurs during the passage of spermatozoa through the caput epididymis and is associated with the migration of the residual cytoplasm from the neck of the spermatozoon to the distal end of the mid-piece.

For the sake of clarity, the morphological features of spermatozoa which are dealt with in this paper are shown diagrammatically in Fig. 1. The spermatozoon is shown to consist of three main components, the *head*, *mid-piece* and *tail*. The head is connected to the mid-piece at the neck. The spermatozoon in Fig. 1a has a fragment of residual cytoplasm attached to the neck; cytoplasmic remnants in this position are here called *neck droplets*. The spermatozoon in Fig. 1b has a fragment of residual cytoplasm attached at the distal end of the mid-piece; cytoplasmic remnants in this position are here called *distal droplets*. Cytoplasmic remnants which are attached at intermediate positions on the mid-piece are arbitrarily classified as neck droplets or distal droplets according to whether they are nearer to the neck or to the distal end of the mid-piece. Fig. 1c shows a spermatozoon with a *broken neck;* here the head is displaced so that its long axis forms an angle with the long axis of the tail. A *disintegrated* spermatozoon is shown in Fig. 1d.

Material and Methods

Material was got from two sterile Guernsey bulls with disintegrated spermatozoa and from three bulls with morphologically normal spermatozoa; one of these three is known to have been normally fertile.

The bulls were shot and bled at the abattoir in the usual way. As soon as practicable after death (within fifteen minutes) the testis and attached epididymides were removed and fixed whole in formal-saline (5 per cent. HCHO; 0.9 per cent. NaCl) containing marble chips. Samples of spermatozoa were taken from incisions made at five different levels of the epididymis (sites 1 to 5). Fluid was pipetted directly off the cut surface or the surface was flushed with formal-saline from a pipette to suspend the spermatozoa and the suspension was pipetted off. The sites for sampling are shown in Fig. 2.

The fixed spermatozoa were examined in wet preparations with the phase-contrast microscope or in smears stained with Heidenhain's iron haematoxylin. Two preparations were made from each site.

Estimates of the proportion of intact spermatozoa, of disintegrated spermatozoa and of spermatozoa with broken necks were made by counting the number of these forms in a total of 200 spermatozoa from each of two preparations from each of three sites (1, 3 and 5). This total of 200 spermatozoa includes heads without tails; tails without heads were not counted.

Estimates of the proportion of spermatozoa with neck droplets and with distal droplets and of spermatozoa without droplets, were also made by counting samples of 200 spermatozoa from each of two preparations at each site. This total of 200 spermatozoa includes tails without heads; heads without tails were not counted.

Results

In the sterile Guernsey bulls the spermatozoa were found to be intact in the most proximal part of the caput epididymis (site 1); in the most distal part of the epididymis (site 5) all the spermatozoa were disintegrated. At intermediate positions (sites 2, 3 and 4) intact spermatozoa, disintegrated spermatozoa and spermatozoa with broken necks were present in varying proportions.

Almost all intact spermatozoa had neck droplets and at the most distal site (site 5) almost all free tails had distal droplets. In most spermatozoa with broken necks, the droplet was at or near the neck and it was clear that the original attachment of the head to the tail was severed, the head being attached to the tail by the attachment of both to the cytoplasmic droplet; some heads were attached to distal droplets. These findings are summarised in Table I and are illustrated in Plate 1.

TABLE I

THE MORPHOLOGICAL FEATURES OF SPERMATOZOA IN SAMPLES TAKEN AT THREE SITES (1, 3 and 5) IN THE CAPUT EPIDIDYMIS FROM TWO STERILE GUERNSEY BULLS *(A AND B) WITH DISINTEGRATED SPERMATOZOA

	Number (per cent.) of intact spermatozoa (i), disintegrated spermatozoa (d) and spermatozoa with broken necks (b)						Number (per cent.) of spermatozoa with neck droplets (n), distal droplets (t) and without droplets (–)					
	i		d		b		n		t		–	
Site	A	B	A	B	A	B	A	B	A	B	A	B
1	86	90	2	6	11	4	100	100	0	0	0	0
3	22	52	11	8	66	40	96	100	1	0	3	0
5	1	0	99	99	0	1	1	2	50	88	49	10

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In Plate 1a the spermatozoa are intact and have neck droplets. Plate 1b shows spermatozoa with broken necks and in these the proximal end of the tail can be seen projecting beyond the droplet. In Plate 1c one spermatozoon is shown with the head fixed to a distal droplet. Plate 1d shows disintegrated spermatozoa from the distal end of the caput epididymis—the isolated tails have distal droplets.

Counts of spermatozoa with neck droplets, with distal droplets and without droplets, in samples taken at three sites from the testis of three bulls with morphologically normal spermatozoa, show that migration of the droplet from the neck to the distal end of the middle-piece also occurs in normal spermatozoa during the course of the journey through the caput epididymis. These findings are summarised in Table II.

TABLE II

THE MORPHOLOGICAL FEATURES OF SPERMATOZOA IN SAMPLES TAKEN AT THREE SITES (1, 3 AND 5) IN THE CAPUT EPIDIDYMIS FROM THREE BULLS (x, y AND z) WITH MORPHOLOGICALLY NORMAL SPERMATOZOA

	The number (per cent.) of spermatozoa with neck droplets (n), with distal droplets (d) and without droplets (–)								
	n			d			–		
Site	x	y	z	x	y	z	x	y	z
1	98	98	98	1	1	1	1	1	1
3	97	99	26	1	0	67	2	0	7
5	17	15	3	62	69	85	21	16	12

Discussion

It has been shown that in Guernsey bulls with disintegrated spermatozoa disintegration occurs in the caput epididymis. Disintegration is apparently associated with the migration of the cytoplasmic droplet from the proximal to the distal end of the mid-piece although the significance of this association is not clear. It is possible that the migrating droplet fails to separate itself from the head and actually tears the head from the mid-piece. It is equally possible that the droplet merely holds the head in position for a time after the normal connections of the head and tail have been severed; if this is the case, there is no clue here to the nature of the real defect. There is no reason to believe that migration of the droplet in the spermatozoa of the sterile bulls differs in any way from that in normal bulls.

Disintegration of spermatozoa commonly occurs as a result of inflammatory changes in the reproductive tract (Lagerlof, 1934) and as a result of various experimental treatments which raise or lower the temperature of the testis. This has been observed in the ram (Phillips & McKenzie, 1934; Gunn, Saunders & Granger, 1942), bull (Lagerlof, 1934; Blom, 1950) and boar (Holst, 1949).

It would be interesting to know if the sequence of events described here occurs when disintegration results from experimental treatment; the evidence on this point (Chang, 1943) is so far inconclusive.

Although previous workers (Branton & Salisbury, 1947) have said that in normal bulls the proportion of spermatozoa with neck droplets is higher in the caput epididymis than in the cauda epididymis, it has not previously been shown that migration of the cytoplasmic droplet normally occurs almost exclusively in the caput epididymis.

Summary

In sterile Guernsey bulls disintegration of the spermatozoa into isolated heads and tails is shown to occur in the caput epididymis.

Disintegration is associated with the migration of the cytoplasmic droplet from the proximal to the distal end of the sperm mid-piece.

In sterile Guernsey bulls migration of the cytoplasmic droplet along the mid-piece occurs while the spermatozoa are passing through the caput epididymis. This is also true of bulls with morphologically normal spermatozoa.

Acknowledgments.—The Guernsey bulls were made available through the co-operation of Mr. S. L. Hignett, B.SC., M.R.C.V.S., Wellcome Veterinary Research Station, Frant; Mr. J. A. Teakle, M.R.C.V.S., Lewes, and Mr. P. S. Hastie, M.R.C.V.S., Ashford. Mr. G. F. Smith, M.R.C.V.S., Beccles, supplied some of the normal material.

References

BLOM, E. (1950). "Om Bedømmelsen af Tyresperma." Mortensen, Copenhagen.

BRANTON, C., & SALISBURY, G. W. (1947). *J. Anim. Sci.* **6**. 154.

CHANG, M. C. (1943). *J. exp. Biol.* **20**. 16.

GUNN, R. M. C., SAUNDERS, R. N., & GRANGER, W. (1943). Bull. Council Sci. Ind. Res. Aust., 148.

HANCOCK, J. L., & ROLLINSON, D. H. L. (1949). *Vet. Rec.* **61**. 742.

HOLST, S. J. (1949). *Nord. Vet.-Med.* **1**. 87.

LAGERLOF, N. (1934). *Acta. Path. Microbiol. Scand Uppsala. Suppl.* **19**.

PHILLIPS, R. W., & MCKENZIE, F. F. (1934). *Mo. Agric. exp. Sta. Res. Bull.* **217**.

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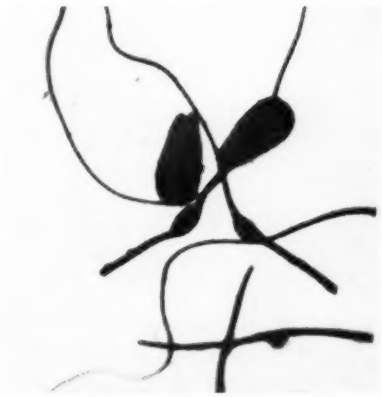
(a)—Intact spermatozoa with neck droplets.

(b)—Spermatozoa with broken necks. The proximal end of the midpiece can be seen projecting from the droplet showing that the head is held in place by the droplet.

(c)—Two spermatozoa are shown. In one the head is attached to a distal droplet.



(d)—Disintegrated spermatozoa. The isolated tails have distal droplets.

Smears of spermatozoa from different levels of the epididymis of a sterile Guernsey bull. Iron haematoxylin. × 1450.

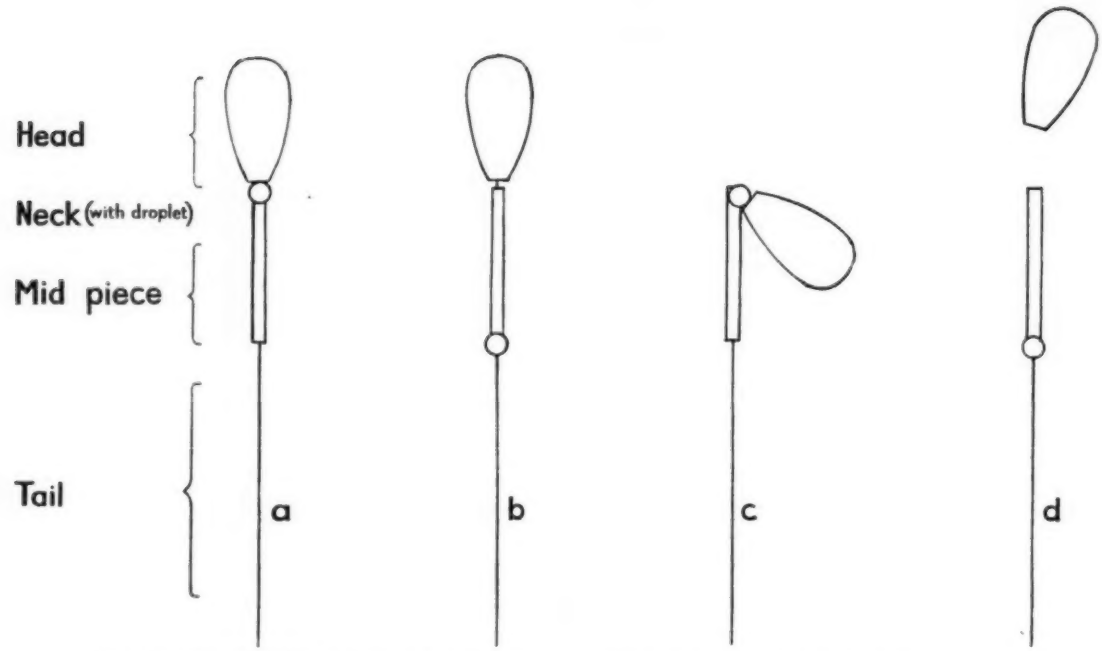


FIG. 1.—The morphological features of epididymal spermatozoa from normal and sterile bulls.

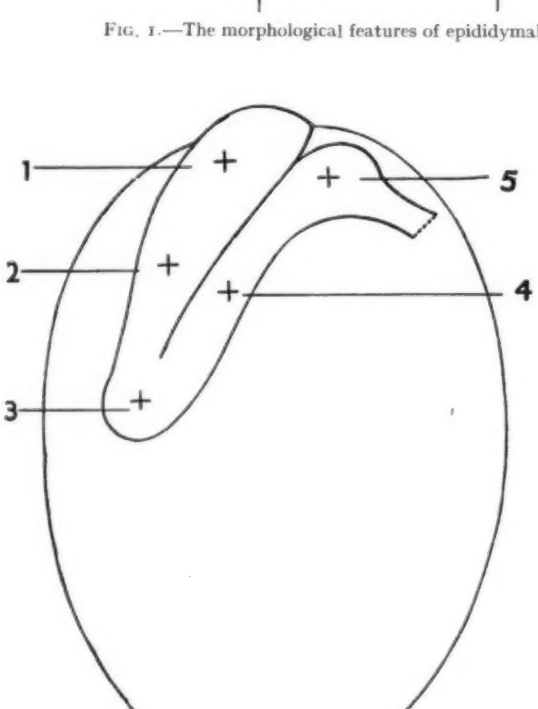


FIG. 1a.—Intact spermatozoon with residual cytoplasm at neck (neck droplet).

FIG. 1b.—Intact spermatozoon with residual cytoplasm at distal end of midpiece (distal droplet).

FIG. 1c. — Spermatozoon with broken neck and attached neck droplet.

FIG. 1d. — Disintegrated spermatozoon —the isolated tail has an attached distal droplet.

FIG. 2.—Diagram of bull testis to show the five sites in the caput epididymis at which samples of spermatozoa were taken for morphological examination (the corpus and cauda epididymis are not shown). (× 1.5.)

"Fluorine Alleviators": A Review

BY

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Introduction

THE term "fluorine alleviator" has been coined to describe substances that can be given to animals in their food with the object of reducing the absorption of fluorine compounds ingested with contaminated herbage. It is generally accepted that fluorine alleviators are principally, if not entirely, of possible value in the alleviation of chronic fluorine poisoning, often known simply as fluorosis.

There are three principal sources of fluorine as a cause of poisoning of livestock: (1) water supplies; (2) contamination from industrial sources; and (3) the use of fluorine containing phosphates as mineral supplements.

Trouble arising from water supplies has been reported from various parts of the world including north Africa, India and Queensland. It has often proved possible to deal with the problem by using other sources of water.

The problem arising from the presence of fluorine compounds in phosphates used as mineral supplements is capable of comparatively easy solution for there are sources of supply from which phosphates with an innocuous fluorine content can be obtained.

It is in connection with fluorine poisoning arising from industrial contamination that a difficult problem has arisen.

The Problem

Sappington (1943) stated that there are at least 28 industrial processes that deliberately or involuntarily discharge fluorine compounds into the atmosphere surrounding factories. The processes involved include aluminium reduction works, brick works, steel works, enamel works, vitreous enamel works and superphosphate works. Electrical power stations may discharge considerable quantities of fluorine into the atmosphere according to the sources of coal and the provision made in the flues and chimneys to trap and extract fluorine and other compounds from the waste products passing into the chimneys.

The literature on fluorosis is literally enormous and it is only necessary for the present purpose to refer to some of the papers dealing with fluorosis in Great Britain. Blakemore *et al.* (1941) described fluorine poisoning of stock in the immediate neighbourhood of brick works in Bedfordshire. Boddie (1945, 1947, and 1948) reported the occurrence of fluorosis around aluminium reduction works in Inverness-shire. (The occurrence of fluorosis in Inverness-shire appears first to have been observed by Cameron in 1937.) Bythell (1947) encountered fluorine poisoning in cattle on fields downwind from an enamel factory; this outbreak was also investigated by the writer of this study. Blakemore *et al.* (1948) reported cases in cattle and sheep in Lincolnshire in an area where ironstone was being calcined with open cast coal. Boddie (1952) established the presence of dental fluorosis in cattle that had been reared in close proximity to a colliery refuse bin that had been burning for over two years. Towers (1954) discussed the occurrence of fluorosis in Yorkshire chiefly in the vicinity of iron and steel works, more particularly those works making basic steel by the open hearth process where fluorspar is used as a flux.

In some factories it may be possible to instal apparatus that will trap the fluorine compounds. This has been done in the chimneys of brick works and some electricity generating stations. In certain factories the cost of installing such apparatus would be out of all proportion to the value of the products being manufactured. Thus in one enamel works the cost of installing the apparatus would have been more than the cost of the whole factory and the interest charges on the cost would have amounted to more than the gross profits earned by the products being manufactured. In respect of aluminium reduction works there are serious technical difficulties in the way of constructing ventilation and extraction machinery that will reduce the emission of fluorine compounds to below a concentration that produces a degree of herbage contamination innocuous to grazing stock. A similar technical difficulty is being experienced in respect of aluminium reduction works in North America.

Then the question of expediency has to be taken into account. In Great Britain many of the factories are producing goods which are being exported to hard currency countries. It is then a matter of considering the relative importance to the country of the deterioration of health of a small number of cattle as compared with the export of manufactured goods to hard currency countries. There is, too, the relative importance as between the employment of 500 or 600 persons in a factory and the comparatively minor damage inflicted on perhaps less than 100 cattle in the area. It must be admitted that these industrial processes must continue and that it may be quite uneconomic, even if it is possible, to control the emission of fluorine compounds into the surrounding atmosphere.

Clinical Manifestations

There are two main clinical types of fluorine poisoning (1) acute and (2) chronic. Practically none of the industrial processes in this country emit sufficient quantities of fluorine compounds to produce acute poisoning. Where that occurs, steps can and should be taken to deal with the nuisance. Chronic fluorine poisoning, often known as "fluorosis," takes two forms, the bone and joint lesions leading to lameness, and the changes in the teeth. The latter are of importance because of the poor wearing qualities of the teeth. The dental changes occur in cattle and

sheep grazing contaminated herbage during the time their permanent teeth are being formed, the bone and joint lesions may occur in any age of cattle or sheep. Horses, though susceptible, appear, compared to cattle and sheep, to be relatively resistant to the effects of fluorosis. In any case with the progress of farm mechanisation there are relatively few horses on the farms involved and these often are aged animals.

The exact degree of herbage contamination that should be regarded as significant cannot be stated with precision for there are seven principal factors that influence the significance.

1. The extent to which the animals are receiving uncontaminated food in addition to contaminated food.

2. The degree to which the animals are restricted to a highly contaminated area in contrast to animals which have an extensive range that allows them in part to graze on lightly contaminated areas.

3. The lack of uniformity in contamination of any area. Wind eddies disperse the fumes in ways that are quite erratic. Animals in one field may be consuming quite considerable quantities of fluorine compound while in those in a neighbouring field the ingestion rate is relatively slow.

4. The composition of the diet particularly as it concerns the calcium content may have some influence on the absorption of fluorine compounds.

5. The emission of fluorine compounds from the factory may be irregular.

6. The dispersal of fumes from the factory is influenced by changes in wind direction and velocity, and also by the rainfall at any given period.

7. The exact composition of the fluorine compounds being discharged into the atmosphere varies according to the manufacturing process involved.

Bearing all these points in mind, it will be realised that figures of herbage contamination only serve as a guide to the extent of damage likely to be caused but are of course an index of the amount of fluorine compounds being discharged into the atmosphere. The figures given below are based on extensive clinical observations coupled with herbage analyses and involve a number of industrial processes (Boddie, 1955). Dental fluorosis has not been observed in cattle and sheep reared on grazings contaminated to the extent of 5 p.p.m. of fluorine in the herbage. Grazing on a pasture contaminated to the extent of 8 p.p.m. 25 per cent. of cattle and sheep developed slight lesions of dental fluorosis. At 14 p.p.m. of fluorine, 100 per cent of cattle and sheep reared in the area showed signs of dental fluorosis. In dairy cows grazing on a pasture contaminated to the extent of 98 p.p.m. bone and joint lesions developed in about one month, lameness disappeared when the cows were removed to a pasture with a fluorine content of 14 p.p.m.

Alleviation Methods

The difficulties mentioned in connection with the removal of the nuisance by modification of the factories have made it necessary to seek other methods of dealing with the problem.

On some farms to prevent dental fluorosis it is possible to remove the young stock to farms remote from the contaminated area where they are reared to the stage of calving heifers. This is usually quite impossible in the smaller type of not too good farm found in industrial areas. An expedient but not a solution for these farms is to give up rearing, and, as their principal business is milk production, to buy in newly calved cows or cows at the drop. This means a radical change from true dairy farming to cow feeding and frequently creates economic difficulties.

The idea therefore developed that it would be useful to look for some substance which when fed to stock would alleviate the effect of continued ingestion of fluorine compounds.

Now fluorine alleviators can usually be furnished to cattle without any difficulty when the cattle, even though out at grazing, are given the antidotal substance mixed with some palatable foodstuff fed in troughs; dairy cattle being brought in twice a day can be fed the alleviator with some concentrate at milking time. Trough feeding of sheep on arable ground presents no difficulty, but the regular administration of an alleviator to hill sheep on free ranch is beset with difficulties which have not yet been solved.

Animals grazing contaminated herbage ingest some fluorine with each mouthful they swallow. Fortunately the mechanism of rumen digestion entails a process of mixing newly ingested food with the rumen contents, removal of foodstuff from the rumen is a gradual process. If then an adequate concentration of the antidotal substance can be built up and maintained in the rumen, ingested foodstuff will come in contact with it as a result of the continuous process of churning that goes on in the rumen. The administration of the antidote twice daily in some foodstuff seems on theoretical grounds sufficient to maintain an adequate concentration in the rumen; it is quite possible that administration once a day would suffice.

In looking for a suitable substance to use as a fluorine alleviator there are some obvious requirements that must be fulfilled. The substance used must be palatable so that animals will consume it readily when mixed with food. Lack of palatability can be overcome to some extent by dilution with food, but obviously if the alleviator requires great dilution it will entail the use of large quantities of food which may not be available or which it may be uneconomic to provide. The substance used must be innocuous even when fed continuously over long periods. Naturally it is desirable that the alleviator should be reasonably effective, but there is every reason for saying it would be improbable that any antidote would prove effective against high herbage contamination in the immediate vicinity of a large factory where the fluorine content of the herbage may attain a figure of 1,000 p.p.m. or more. On the other hand a fluorine alleviator that was effective, though not necessarily completely effective, against herbage concentrations of 100 p.p.m. would be of great assistance in many areas where industrial fluorosis is a problem. Even if a fluorine alleviator were only 50 per cent. effective—*i.e.* depressed the absorption and retention of fluorine compounds by one half—it would be of great assistance in mitigating the effect on the teeth of growing stock and would in many instances virtually eliminate lameness.

Literature on Antidotes to Chronic Fluorine Toxicity

Though as has been stated the literature on fluorosis is literally enormous, there is comparatively little on the question of trying to counteract the effects of continued ingestion of fluorine compounds.

Marcovitch & Stanley (1942) state that the most commonly accepted antidote for fluorides is calcium in some form. In their experimental work they based their findings on weight gain. They fed sodium fluoride to rats at the rate of 2,000 p.p.m. in the diet. When " hydrated lime " was added to the diet there was a 49 per cent. gain in weight compared with the controls which had no antidote. When aluminium sulphate was added to the diet at the rate of 4,000 p.p.m. there was a weight gain of 68 per cent. The fluorine content of these diets was very high and the results tend to suggest that toxic actions were being produced which were more general in effect than those usually seen in cattle and sheep.

Lawrenz & Mitchell (1941) stated that they used rats and a diet containing 9 p.p.m. of fluorine and 0.73 per cent. of calcium compared with a diet containing 9 p.p.m. of fluorine and 0.23 per cent. of calcium in which the calcium-phosphorus ratios were 1.40 to 1 and 0.44 to 1 respectively. The retention of fluorine, as measured, by bone analysis, was depressed in the higher calcium diet from 10.5 to 12.8 per cent. as compared with the lower calcium diet.

Majumdar *et al.* (1943) used adult healthy hill bulls, three to five years old, weighing 250 to 300 lb. in groups of three for experimental purposes. Sodium fluoride fed at a level of 3 to 4 mg. of fluorine per kg. bodyweight produced signs in cattle comparable to those identified as being due to fluorosis. One group received unstated quantities of a calcium supplement. It is stated that this mitigated the symptoms of poisoning and lowered the fluorine content of the bones and teeth. Presumably the symptoms of poisoning were those of bone and joint fluorosis.

Majumdar & Ray (1946) claimed that in experiments with hill bulls the addition of calcium compounds to the diet was only effective in protecting animals against fluorine intoxication when it is required to bring the calcium-phosphorus ratios to optimum levels. In the summary of this paper it is stated that the ingestion of aluminium sulphate was very effective in preventing fluorosis but in the protocols of the experiment it is stated that aluminium phosphate was given to the animals in the aluminium group.

Mitchell & Edman (1952) in a review of the fluorine problem in livestock feeding point out that the one exception to favourable results with calcium supplements, namely that of Majumdar & Ray, may have been due to accentuation of a phosphorus deficiency rather than an intensification of fluorine toxicity.

Sharpless (1936) described experiments in rats in which a diet containing 1,000 p.p.m. of sodium fluoride retarded growth, but if 0.056 per cent. of aluminium was added to the diet growth was normal. One per cent. calcium carbonate gave some protection but if 2 per cent. was added, growth was normal. Studying the effect of fluorine on the teeth of rats, Sharpless showed that 0.5 per cent. aluminium chloride would retard changes for five to six weeks, but 2 per cent. aluminium chloride retarded the changes for six months.

A combination of 2.0 per cent. calcium carbonate with 2.0 per cent. aluminium chloride was effective in preventing dental fluorosis in rats in so far as the naked eye appearances of the teeth were concerned.

Sharpless suggests that since aluminium compounds are thought not to be absorbed from the intestine, it is assumed that the protective action takes place in the intestine probably by the formation of a compound of aluminium and fluorine which is very slightly disassociated.

Venkataramanan & Krishnaswamy (1949) carried out experiments with rats extending over 15 weeks. There were three groups, one received a normal diet, the second group received 1 mg. of fluorine of sodium fluoride in the diet (presumably daily) and the third group received 1 mg. of fluorine as sodium fluoride and 200 mg. of aluminium sulphate. There was no appreciable differences in weight gains between the three groups. At the end of the experiment the rats were sacrificed and the fluorine content of the femur estimated. In the control group the fluorine content of the bones was 1,140 p.p.m.; in the group receiving fluorine alone the content was 3,810 p.p.m. and in the group receiving fluorine plus aluminium the content was 3,120 p.p.m. The number of rats involved is not stated, nor are the range in figures of the respective groups given.

Becker *et al.* (1950) fed young rats for six weeks on diet containing 13 to 445 p.p.m. fluorine as sodium fluoride. The fluorine content of the tibia and femur was used to indicate the degree of fluorine toxicosis. Aluminium sulphate fed as 0.53 per cent. of the diet caused a 45 per cent. (30-57) diminution of the fluorine content of the bones. Aluminium chloride was stated to be of equal value in alleviating chronic fluorosis. Alumina was not considered to be so effective as the sulphate or chloride.

Experiments with sheep indicated that aluminium served to diminish the absorption of fluorine from the intestinal tract.

Anderson (1955) has commented on the difficulty of getting animals to consume aluminium sulphate and aluminium chloride because of the distasteful nature of the compounds.

Discussion

It will be seen that this review of the literature tends to confirm the statement by Marcovitch & Stanley (1942) that calcium in some form has been accepted most commonly as an antidote to fluorine. There is, however, good evidence that aluminium compounds are useful, perhaps more useful than calcium compounds. In considering the relative efficacy of the aluminium compounds, the sulphate, the chloride and the oxide, it is necessary to consider not only the direct evaluation of the alleviator effects but also the palatability of the individual compound. On this score there seem to be grounds for preferring the oxide. Then some consideration must be given to the possible long term effects of prolonged feeding of the sulphate and chloride; there do not seem to

be any reasons for anticipating that prolonged feeding of aluminium oxide would have any deleterious effect.

The information to be gleaned from this review of the literature is largely applicable to the short term effect of feeding fluorine antidotes and the information thus furnished suggests that bone and joint fluorosis might be mitigated or even prevented by the use of a suitable antidote during the normal summer grazing season in this country. The literature quoted does not give much of a guide as to the possible value of fluorine antidotes to prevent bone and joint fluorosis in animals grazing contaminated pastures for long periods such as a year or more, and it does not clearly establish the value of antidotes in the prevention of dental fluorosis in young stock grazing continuously on contaminated pastures from birth—such as for instance suckled calves and lambs.

The information thus available suggests that there are reasonable prospects of using substances as fluorine alleviators that could be fed to cattle or sheep when mixed with ordinary foodstuffs. It is naturally desirable that experiments should be carried out in cattle and sheep. There are, however, quite serious practical difficulties in the way of carrying out such experiments on ordinary farms. Experiments carried out for a short period of time could give quite misleading results, since there are substantial fluctuations in the severity of the herbage contamination. These variations may be due to irregular emission of fumes from the factory, and weather conditions, both as regards rainfall and prevalence of wind, which influence the precipitation and dispersal of fluorine compounds and also affect the rate of growth of the herbage. Then there is the problem of choosing a locus. Where only one factory is involved it may be possible closely to correlate the factory losses of fluorine compounds with the effects produced on stock. Where several factories are involved there may be no possibility of establishing such a correlation but the probability is that the herbage contamination will be more consistently uniform. Long term experiments may put a considerable strain on the farm staff and any mix-up of the experimental animals could vitiate the entire affair. Then it is essential that there should be control animals, but here there are two difficulties, one is that often the animals available would be insufficient to produce two groups of adequate size and secondly that the owner naturally feels that if the fluorine alleviator has any value he would like all his cattle, or sheep, to benefit.

It will, however, be realised that if an experiment were conducted without controls and only extended over a short period of time, it might be quite impossible to assess the results.

Summary

The problem of industrial fluorosis in cattle and sheep is described and discussed.

The clinical manifestations are described.

Possible methods of alleviation are considered, including the use of fluorine alleviators.

The literature on fluorine antidotes is reviewed and discussed.

References

ANDERSON, R. W. (1954). Personal communication.

BECKER, D. E., GRIFFITH, J. M., HOBBS, C. S., & MACINTIRE, W. H. (1950). *J. Animal Sci.*, **9**. 647.

BLAKEMORE, F., BOSWORTH, T. J., GREEN, H. H., & MURRAY, M. (1941). *Proc. Roy. Soc. Med.* **34**. 391.

———, ———, & ———. (1948). *J. Comp. Path.* **58**. 267.

BODDIE, G. F. (1945). *Proc. Nutr. Soc.* **3**. 94.

———. (1947) *Vet. Rec.* **59**. 301.

———. (1948). *Med. Res. Coun. Mem.* No. 22.

———. (1952). Unpublished observation.

———. (1955). Unpublished observations.

BYTHELL, D. W. P. (1947). Personal communication quoted by Boddie, G. F. (1947). *Vet Rec.* **59**. 470; quoted by Blackmore *et al.* (1948). *J. Comp. Path.* **58**. 267.

CAMERON, J. (1937). Personal communication.

LAWRENZ, M., & MITCHELL, H. H. (1941). *J. Nutrition.* **22**. 91.

MAJUMDAR, B. N., RAY, S. N., & SEN, K. C. (1943). *Indian J. Vet. Sci.*, **13**. 95.

———. (1946). *Ibid.* **16**. 107.

MARCOVITCH, S., & STANLEY, W. W. (1942). *J. Pharmacol. Exptl. Therap.* **74**. 235.

MITCHELL, H. H., & EDMAN, MARJORIE. (1952). *Nutr. Abstr. Rev.* **21**. 787.

SAPPINGTON, C. O. (1943). Essentials of Industrial Health, Philadelphia.

SHARPLESS, G. R. (1936). *Proc. Soc. Exp. Biol. Med.* **34**. 562.

TOWERS, K. G. (1954). *Vet. Rec.* **66**. 355.

VENKATARAMANAN, K., & KRISHNASWAMY, K. (1944). *Ind. Jour. Med. Res.* **37**. 277.

NATIONAL SHEEP BREEDERS' ASSOCIATION

During the week ending October 22nd, 1955, the N.S.B.A. met at Windermere. A highlight of the conference was a paper read by Mr. J. F. Robinson of the Hill Farming Research Organisation, Edinburgh.

Referring to hill sheep, Mr. Robinson pointed out that scarcity of food during the winter generally leads to loss of bodyweight, and that pregnancy normally coincides with this period. This may be the cause of low lamb crops. Much of the work of the Hill Farming Research Organisation is to be devoted to investigating the best method of feeding hill sheep.

A MISSING VOLUME

THE VETERINARY RECORD, Vol. 47 (1935), is missing from the R.A.V.C. Library, Aldershot. Will the borrower kindly return, or would any Library or Institution donate a spare volume of this year and number, if in possession of one.

News and Comment

SHERRY PARTY AT CAMBRIDGE

On Wednesday evening, October 19th, the day preceding the official opening of the new Veterinary School at Cambridge, the President of the Royal College of Veterinary Surgeons, Mr. Sumner, and Mrs. Sumner, held a sherry party at the Garden House Hotel, Cambridge, for the Professors, Readers, Lecturers and other staff of the School and their ladies. Among those present were Sir Lionel and Lady Whitby, Lt.-Col. and Mrs. Lasher (representing the American Veterinary Medical Association), the Presidents and Honorary Secretaries of the Eastern Counties, Essex, and Hertfordshire and Bedfordshire Divisions of the British Veterinary Association and their ladies, Mr. and Mrs. Howard Dawes, and Mr. and Mrs. H. I. Field.

PERSONAL

Births

BRUNSDON.—On October 16th, 1955, at Butleigh Hospital, Somerset, to Jean, wife of J. R. Brunsdon, M.R.C.V.S., a son, Paul Richard.

LEAM.—On October 7th, 1955, at the Princess Margaret Hospital, Nassau, Bahamas, to Mavis, wife of Gordon Leam, B.V.SC., M.R.C.V.S., a daughter, Deborah Anne.

STEEL.—On October 17th, 1955, at The Barratt Maternity Home, Northampton, to Betty, wife of E. R. Steel, M.R.C.V.S., 61, Derngate, Northampton, a son, Philip Edward.

WILLIAMS.—On October 15th, 1955, at Andover War Memorial Hospital, Hampshire, to Patricia, wife of J. R. L. Williams, M.R.C.V.S., a son, James Julian, brother for Jane.

Marriage

STEWART—FENTON.—On October 15th, 1955, at All Saints Church, Peppard, Oxon, David Little Stewart, M.R.C.V.S., son of the late Mr. and Mrs. Alexander Stewart, Millport, Isle of Cumbrae, to Helen Patricia Fenton, younger daughter of Mrs. H. G. Fenton, Peppard, Oxon.

COMING EVENTS

October

29th (Sat.). Autumn Meeting of the Scottish Metropolitan Division, B.V.A., at the Scottish Milk Marketing Board, Newlands Cattle Breeding Centre, Perth, 2.45 p.m.

First Annual General Meeting of the Veterinary Officers' Section of the Ministry of Agriculture Branch of the Institution of Professional Civil Servants in the Auditorium of the Wellcome Research Institute, 183-193, Euston Road, London, N.W.1, 2 p.m.

November

3rd (Thurs.). General Meeting of the Central Division, B.V.A., at the Royal Veterinary College, Camden Town, N.W.1, 5.30 p.m.

4th (Fri.). The Annual Dinner and Dance of the Royal Counties Division, B.V.A., at the Chequers Hotel, Newbury, 7.30 p.m.

8th (Tues.). Ordinary General Meeting of the Society of Practising Veterinary Surgeons at the Bell Hotel, Leicester, 2.30 p.m.

9th (Wed.). Meeting of the A.V.T. & R.W.'s, Scottish Regional Group, in the Royal (Dick) School of Veterinary Studies, 2.15 p.m.

Ordinary General Meeting of the Sussex Division, B.V.A., at the Old Ship Hotel, Brighton, 2.30 p.m.

General Meeting of the Lancashire Division, B.V.A., in the University Veterinary Hospital, Pembroke Gardens, Liverpool, 2.30 p.m.

11th (Fri.). General Meeting of the Yorkshire Division, B.V.A., in the Hotel Metropole, Leeds, 2.30 p.m.

15th (Tues.). General Meeting of the South-east Midlands Division, B.V.A., at Franklin's Garden Hotel, Northampton, 8 p.m.

17th (Thurs.). Annual Dance of the Victoria Veterinary Benevolent Fund, County of Ayr, Ladies Guild at Western House, Ayr.

General Meeting of the Midland Counties Division, B.V.A., at the Birmingham Medical Institute, 154, Great Charles Street, Birmingham, 2.30 p.m.

General Meeting of the South Eastern Division, B.V.A., at the Royal Star Hotel, Maidstone, 2.30 p.m.

COUNCIL AND COMMITTEE MEETINGS IN LONDON

Wednesday, November 2nd

10.30 a.m.—Veterinary State Medicine Committee.

2.00 p.m.—Parliamentary and Public Relations Committee.

3.15 p.m.—Home Appointments Committee.

Thursday, November 3rd

10.30 a.m.—Organising Committee.

2.00 p.m.—General Purposes and Finance Committee.

Friday, November 4th

10.30 a.m.—Council.

R.C.V.S. EXAMINATIONS

November 4th, Friday.—Pharmacology, etc. Examination (Revised Syllabus). Written Examination.

November 7th, Monday.—Pharmacology, etc. Examination (Revised Syllabus). Oral and Practicals (London and Dublin).

November 9th, Wednesday.—Animal Management Examination, Dublin.

December 8th, Thursday.—M.R.C.V.S. Written Examinations.

December 9th, Friday.—M.R.C.V.S. Written Examinations.

December 12th, Monday.—M.R.C.V.S. Practical and Orals commence (Panels " A " and " B ").

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Angus. Kinpurnie Farm, Newtyle (Oct. 19).

Ayr. Sidehead Farm, Hurlford, Kilmarnock (Oct. 18); Low Todhill Farm, Kilmarnock (Oct. 24).

Cheshire. Brayne Hall, Worleston, Nantwich (Oct. 21).

Derby. Griff Grange Farm, Brassington (Oct. 19); Rodsley Court Farm, Rodsley, Ashbourne (Oct. 22); Alton Hall Farm, Kirk Ireton (Oct. 24).

Lancs. Landslide Farm, Leigh (Oct. 21); Tarnwater Farm, Tarnwater Lane, Ashton-with-Stodday, Lancaster (Oct. 24).

Renfrew. Priestside Farm, Kilmacolm (Oct. 24).

Salop. Newtown Farm, Much Wenlock (Oct. 18).

Staffs. Lower Birche's Farm, Rugeley (Oct. 19); Glebe Farm, Hanbury, Burton-on-Trent (Oct. 20); Wormhill Farm, Rushton (Oct. 21); Hobbergate Farm, Old Road, Oulton Heath, Stone (Oct. 22); Doglands Farm, Fradswell; Eccleshall Road Farm, Muckleston, Market Drayton; Lower Birches Farm, Densford Road, Rugeley (Oct. 24).

Fowl Pest

Essex. Moorcroft Smallholding, Moorcroft Avenue, Thundersley (Oct. 21).

Lancs. Hillock Farm, Knoll Lane, Little Hoole, Preston; Rivington House, Marsh Lane, Longton, Preston; Norris House, Marsh Lane, Longton, Preston; Breskens, Hall Lane, Longton (Oct. 19); Taylors Farm, Marsh Lane, Longton; Nooklands, Marsh Lane, Longton (Oct. 20); Blackhurst Farm, Marsh Lane, Longton (Oct. 21).

Norfolk. Clint Green Farm, Yaxham, Dereham; Rosebank, Norwich Road, Clint Green, Dereham (Oct. 18); 78, Ferrier's Allotments, Cobholm, Gt. Yarmouth; Woolpack Public House, Norwich Road, Yaxham, Dereham (Oct. 19); Boundary Fruit Farm, Ash Lane, Carleton Road, Norwich (Oct. 20); Chapel Street, New Buckenham, Norwich; Kings Street, New Buckenham; Pear Tree Farm, Welborne, East Dereham (Oct. 22); Sneath Farm, Moulton St. Michael, Norwich (Oct. 23); White House, Yaxham, Dereham; Ash Farm, Carleton Rode, Norwich; Shubbery Farm, Carleton Rode, Norwich (Oct. 24).

Oxford. Church Lane, Weston-on-the-Green, Bicester; Oxford House, Weston-on-the-Green; Church Lane and The Forge, Weston-on-the-Green (Oct. 22).

Swine Fever

Gloucs. Piggeries, Collin Lane, Willersley, Broadway (Oct. 19).

Pembroke. Stacpole Quay Farm, Stacpole (Oct. 24).

Salop. Manor Farm, Preston Gubbalds (Oct. 19).

Staffs. Marsh & Baxter Emergency Slaughter House, North Street, Brierley Hill (Oct. 19); High House Farm, Station Road, Great Wyrley, Walsall (Oct. 24).

Stirling. Glenyards Farm, Bonnybridge (Oct. 24).

Warwicks. Stone Cottage Farm, Fillongley, Coventry; Manor Farm, Wroxall (Oct. 19); The Nuthurst, Shrewley (Oct. 24).

Yorks. Church Farm, Main Street, Tockwith (Oct. 22); 49, Lime Tree Lane, Bilton, Hull (Oct. 24).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A

OPENING OF CONGRESS

Sir,—May I suggest that a brief interval be observed at the beginning of our annual Congress so that we can each privately pray for God's guidance in our work and for His Blessing upon ourselves, our colleagues and our profession.

It seems a sad and rather surprising fact that, at B.V.A. Congresses, there is no set official opportunity for commending our work to God's Providence.

Yours faithfully,

BRENDAN HALPIN.

20, Passey Place,
Eltham, S.E.9.

October 24th, 1955.

FEES FOR INSURANCE INSPECTION

Sir,—The point raised in Mr. Joss's letter in THE RECORD, dated October 15th, 1955, should arouse the indignation of members of our profession. The scale of fees for insurance work has been an insult to us for many years, and it is high time that the matter received some publicity, if not action.

In 1953 I wrote to the General Secretary of the B.V.A. on this matter, but was informed that there was little likelihood of improving the present scale, which was fixed with the Tariff Companies in 1945. I then raised the matter at a local divisional meeting of the B.V.A., but my proposition was not received with much enthusiasm.

There should be no necessity to list the reasons for an increase in our fees, as these must be obvious to any thinking veterinary surgeon.

Yours faithfully,

RICHARD H. C. PENNY.

The Black Lion Yard,
High Street,
Leighton Buzzard,
Beds.

October 18th, 1955.